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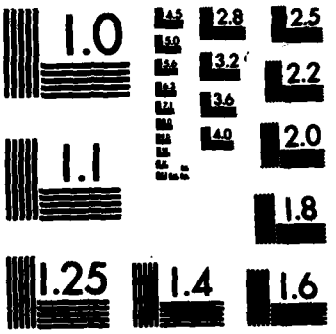

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Thermoregulatory responses were examined in five male subjects during cycle ergometer exercise (50% of maximal aerobic power) in a temperate (T_a=28°C, rh=30%) environment. These exercise tests were conducted following normal sleep (control) and after 33 h of wakefulness. In comparison to control levels, sleep deprivation resulted in a greater increase (26%) in esophageal temperature from rest to final exercise values; sleep deprivation also resulted in reduced total body (27%) and local (19%) sweating rates as well as lowered sweating rate sensitivity (38%). An asynchronous rather than synchronous

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sweating rate pattern was frequently observed during the sleep deprivation test. Following sleep deprivation the threshold (0.11°C) and sensitivity (42%) for chest heat conductance were decreased. We conclude that sleep deprivation depresses thermoregulatory responses to physical exercise and that these responses may be mediated by the central nervous system.

HUMAN RESEARCH

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

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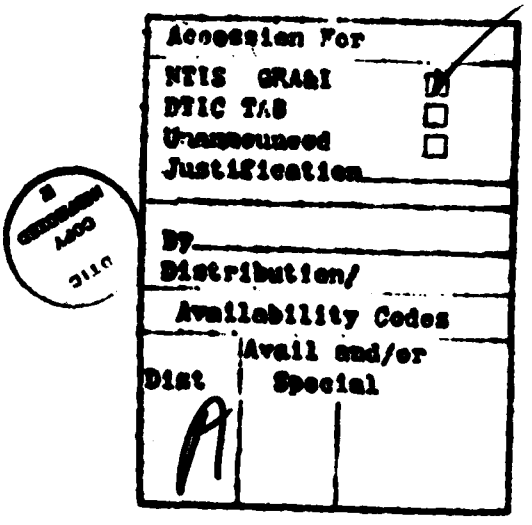

Sleep Deprivation Depresses Thermoregulatory Responses to Physical Exercise

Michael N. Sawka[1] and Richard R. Gonzalez[2]

[2]John B. Pierce Foundation Laboratory and
Yale University School of Medicine,
290 Congress Avenue, New Haven, CT 06519

and

[1]US Army Research Institute of Environmental Medicine,
Kansas Street, Natick, MA 01760

Abstract

Thermoregulatory responses were examined in five male subjects during cycle ergometer exercise (50% of maximal aerobic power) in a temperate (T_a = 28°C, rh = 30%) environment. These exercise tests were conducted following normal sleep (control) and after 33 h of wakefulness. In comparison to control levels, sleep deprivation resulted in a greater increase (26%) in esophageal temperature from rest to final exercise values; sleep deprivation also resulted in reduced total body (27%) and local (19%) sweating rates as well as lowered sweating rate sensitivity (38%). An asynchronous rather than synchronous sweating rate pattern was frequently observed during the sleep deprivation test. Following sleep deprivation the threshold (0.11°C) and sensitivity (42%) for chest heat conductance were decreased. We conclude that sleep deprivation depresses thermoregulatory responses to physical exercise and that these responses may be mediated by the central nervous system.

Moderate physical exercise following acute sleep deprivation is common among individuals in a variety of occupations. Previous investigations indicate that sleep deprivation has minimal effects on the bioenergetic and cardiovascular responses of individuals during physical exercise (1,2,3). However, the effects of sleep deprivation on thermoregulatory responses during physical exercise have not been examined. Sleep deprivation might alter thermoregulatory responses since a circadian pattern has been established for sweating rate and peripheral blood flow responses for individuals at rest (4,5) and during physical exercise (6). In fact, sleep deprivation has been documented to reduce an individual's oral temperature at rest (3).

The present investigation demonstrates that sleep deprivation reduces man's ability to dissipate metabolic heat during moderate physical exercise. This is manifested by a reduced sensitivity for local sweating rate and chest heat conductance. In addition, after sleep deprivation an asynchronous, rather than a normal synchronous, local sweating rate response was frequently observed.

Five male volunteers with a peak oxygen uptake ($\bar{X} \pm sd$) of 52 ± 2 ml $\cdot$ kg^{-1} $\cdot$ min^{-1} and a normal daily sleep duration of 8.2 ± 0.2 h participated in this study. Prior to experimental testing, subjects were screened for sleep habits, and familiarized with the test procedure. Subjects completed a practice, control (C) and sleep deprivation (SD) exercise test. The practice test was employed to control for habituation. The practice and C tests were preceded by two nights of normal sleep, while the sleep deprivation test was preceded by 33 h of wakefulness. Subjects were monitored and instructed to remain inactive during the sleepless night. Each subject followed similar activity and dietary regimens for the 48 h period preceding the C and SD tests.

All exercise tests were conducted at 1500 h in a temperate ($T_a = 28^{\circ}C$, rh = 30%) environment. The tests consisted of rest for 20 min and cycle

ergometry at 50% of peak oxygen uptake for 40 min. Physiological measurements included: esophageal temperature at heart level, skin temperature by thermocouples, local sweating responses by dew point hygrometry (7) and chest heat conductance by heat flow discs (8). In addition, oxygen uptake was determined by open circuit spirometry.

Each subject completed the entire 40 min of exercise for the C and SD tests. During both exercise bouts a metabolic rate of 620 ± 55 W was elicited. Esophageal temperature was not significantly different between the C and SD test when at rest or at the end of exercise. However, the increase in esophageal temperature from rest to the end of exercise was $0.53 \pm 0.15^{\circ}C$ and $0.67 \pm 0.14^{\circ}C$ for the C and SD tests, respectively. This represented a 26% greater increase ($P = 0.08$) in esophageal temperature during the SD test. Mean skin temperature remained constant at $32^{\circ}C$ during both the SD and C tests. Total body sweating rate, as determined by change in body weight, was 27% ($P < 0.01$) less during the SD (224 ± 16 $g \cdot m^{-2} \cdot h^{-1}$) than during the C ($307 \pm 15$ $g \cdot m^{-2} \cdot h^{-1}$) test. Final exercise local sweating rate (d_s) was 19% ($P < 0.01$) less during the SD (0.79 ± 0.23 $mg \cdot cm^{-2} \cdot min^{-1}$) than during the C ($0.98 \pm 0.16$ $mg \cdot cm^{-2} \cdot min^{-1}$) test. In addition, during the SD test an asynchronous rather than normal synchronous d_s pattern was observed for three of the subjects. Asynchrony was not observed during any practice or control tests. Final exercise chest heat conductance was 10% ($P < 0.05$) less during the SD (45 ± 6 $W \cdot m^{-2} \cdot {}^{\circ}K^{-1}$) than during the C ($50 \pm 6$ $W \cdot m^{-2} \cdot {}^{\circ}K^{-1}$) test. Table 1 presents the threshold and sensitivity data for local sweating rate and chest heat conductance. Chest heat conductance threshold was $0.11^{\circ}C$ ($P < 0.05$) lower during the SD test. Decreases of 38% ($P < 0.01$) and 42% ($P < 0.05$) were observed for local sweating rate sensitivity and chest heat conductance sensitivity during the SD as compared to the C test.

The marked reductions in evaporative and dry heat loss following sleep deprivation would be expected to have a more profound effect on esophageal temperature in hotter and more humid environments. The present study's temperate environment was selected to insure that subjects would not discontinue exercise because of thermal strain. The mechanism(s) for the decreased total body and local sweating rates following sleep deprivation remain unclear. However, these reductions could result from changes mediated by the central nervous system (CNS) and/or by peripheral changes at the sweat gland. The following supportive evidence may favor the former mechanism in explaining the depressed sweating rate.

During the C test, a normal synchronous pattern for sweat secretion was always observed. Following sleep deprivation, however, an asynchronous pattern was observed in three subjects. This asynchrony suggests a disruption in the pattern of neural activity received from the hypothalamic thermoregulatory center (9). If local sweating rates had been reduced along with a synchronous discharge, the mechanism(s) could more likely have been peripheral at the sweat gland. Likewise, chest heat conductance was also depressed during the SD test. These heat conductance changes probably reflect a reduced cutaneous blood flow (10), mediated by changes in vasomotor tone. Apparently, sympathetic vasoconstrictor tone was attenuated at a lower esophageal temperature, but the maximal level of cutaneous vasodilation was reduced following sleep deprivation. Therefore, both the asynchronous sweat secretion and the reduced chest heat conductance responses suggest a CNS mediated change in thermoregulatory responses.

Sleep deprivation may alter the output signal from the hypothalamic thermoregulatory center, or modify the signal after its initiation. The altered output signal could result from a depressed hypothalamic thermoregulatory

center. The hypothalamus could have been depressed as a result of a) changes in the concentration of monoamines and/or b) reduced afferent input from the ascending reticular activating system. Monoamines such as 5-hydroxytryptamine and norepinephrine have been shown to affect thermoregulatory responses (11) as well as to influence sleep-wake cycles in mammals (12). Also, the excitatory neurons of the reticular activating system may fatigue following prolonged wakefulness and thereby reduce the activation of the hypothalamus (12). As a result, for a given thermal drive, the hypothalamus would be desensitized and less able to initiate the effector signal. Another possibility for the depressed thermoregulatory responses is that the effector signal was altered after initiation in the hypothalamus. A depression of descending reticular tract activity could result in the observed asynchronous d_s recordings and reduced responses.

While additional research is needed to elucidate the physiological mechanism(s), we have demonstrated that sweating rate and chest heat conductance are altered during moderate intensity exercise following sleep deprivation. These findings have important implications for individuals performing exercise in thermally stressful environments while sleep deprived.

Michael N. Sawka, Ph.D.
US Army Research Institute of
Environmental Medicine
Kansas Street
Natick, MA 01760

Michael N. Sawka 12-21-92

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Table 1. Threshold and Sensitivity of Local Sweating Rate (d_s) and Chest Conductance (K_{ch}) Responses.

	Local Sweating Rate		Chest Conductance	
	Threshold (^oC)	Sensitivity ($\Delta d_s \cdot \Delta T_{es}^{-1}$)	Threshold (^oC)	Sensitivity ($\Delta K_{ch} \cdot \Delta T_{es}^{-1}$)
Control				
$\bar{X}$	37.10	1.95	37.25	41.83
$\pm$sd	0.17	0.59	0.21	11.60
Sleep Deprivation				
$\bar{X}$	37.13	1.20	37.14	24.28
$\pm$sd	0.19	0.23	0.26	6.09
%Δ	0	-38	0	-42
P	NS	< 0.01	< 0.05	< 0.05

Sweating rate is in $mg \cdot cm^{-2} \cdot min^{-1}$ and chest conductance is in $W \cdot m^{-2} \cdot {}^oK^{-1}$; ΔT_{es} is change in esophageal temperature from rest to the end of exercise.